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# CARDIOVASCULAR AND METABOLIC RECOVERY OF A MARATHON RUNNER[1]

M. N. SAWKA, D. S. MILES and R. M. GLASER, Department of Physiology, Wright State University, School of Medicine, Dayton, OH 45435
R. G. KNOWLTON and J. B. CRITZ, Southern Illinois University, Carbondale, IL 62901

*Abstract.* Physiological recovery after a competitive marathon race was studied in a trained 43 year old male. The subject completed a progressive intensity treadmill test to exhaustion 6 days prior to, and 1, 3, 5, and 9 days post-marathon. Post-marathon values of cardiac output, stroke volume, heart rate and oxygen uptake during submaximal exercise were similar to pre-marathon values. Maximal blood lactate concentrations were decreased from pre-marathon levels by 50%, 58%, and 26% during the first week of recovery. Maximal blood lactate concentration, however, again achieved the pre-marathon level after 9 days of recovery. This concentration was believed to reflect the reestablishment of muscle glycogen stores comparable to pre-marathon levels. It was concluded that cardiovascular function demonstrated little after effects from the marathon, but at least one week was required for full metabolic recovery.

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Marathon races (26.2 miles) are one of the most stressful athletic events encountered by the human body (Milvy 1977). During these races, athletes compete at metabolic intensities exceeding 15 Kcal·min$^{-1}$ for time periods that may exceed 3 hours. Numerous investigations have focused attention on the physiological responses during these events (Costill and Fox 1969, McKechnie *et al* 1967, Milvy 1977), but little data have been published on the subsequent recovery period. This paucity of data concerning physiological implications of recovery prompted the present investigation. The purpose of this study was to quantitate metabolic and cardiovascular recovery of a 43 year old male runner for several days following competition in the St. Louis Marathon.

## METHODS

The subject was a healthy 43 year old male, 187 cm in height, 75.5 kg in weight, with 9.8% body fat (as estimated by $K^{40}$ technique) and had a maximal oxygen uptake of 56 ml·kg$^{-1}$·min$^{-1}$. Pre-marathon training consisted of running 70 miles per week at approximately 8 mph. During the 2 months preceding the race, the subject had 15 runs of at least one half the marathon distance. In addition, the subject accumulated an annual running distance of 2,700 miles.

Physiological measurements were obtained during an incremental intensity, continuous treadmill protocol (McDonough and Bruce 1969) that was preceded by a 5 min warm up walk. A reference pre-marathon treadmill test was given six days prior to the marathon and subsequent tests were completed 1, 3, 5, and 9 days post-marathon. The subject also completed four maximal treadmill runs prior to the reference pre-marathon test. We used these 4 runs to control for habituation and collected no data at that time. We conducted all tests during the morning in an air-conditioned laboratory at 22 ° ±1 °C and 50% relative humidity.

Oxygen uptake was determined at each stage of the treadmill test. Samples of respiratory gases were analyzed with a carbon dioxide analyzer (Godart Capnograph) and oxygen analyzer (Beckman OM-11) calibrated against reference gases of known concentrations (Micro-Scholander technique). We measured ventilation with a Parkinson-Cowan dry gas meter calibrated against a 350 liter Tissot gasometer. Cardiac output was determined by the carbon dioxide rebreathing technique at each stage through the fifth workload as previously described (Knowlton and Adams 1974). Values for cardiac output determined by this method have been reported during maximal exercise (Ashton and McHardy 1963, Jernerus *et al* 1963, Miyamura and Honda 1972); however, one of the assumptions of this method is that the subject be in a steady-state. Because it was apparent that our subject was not in a steady-state after the fifth stage, we have reported

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cardiac output values for the first 5 stages only. Heart rate for the calculation of stroke volume was derived from the ECG. We took finger capillary blood 5 minutes post-exercise and determined blood lactate by an enzymatic method (Boehringer Mannheim reagents).

## RESULTS AND DISCUSSION

During the marathon race, the shaded dry bulb temperature ranged from 13 °C to 22 °C with a mean relative humidity of 46%. The running course was generally unprotected from radiant heat. The subject completed the race in 3 hours and 17 min, though he ran the same course 28 min quicker during the previous year. Since the normal temperature for this date is 8 °C, we felt the unseasonable high temperature during the studied race produced a greater thermal stress on the subject and resulted in a decreased performance.

The subject did not train during the studied 9 day recovery period. During this time, he gradually improved in his ability to sustain treadmill running (table 1). Although habituation may have contributed to the increase in run time, it was unlikely since the subject has had considerable treadmill experience. It appears that rest from training and gradual physiological recovery may account for this improved performance.

The cardiovascular variables demonstrated the expected responses to graded exercise (table 1). Cardiac output approached 30 $\ell \cdot min^{-1}$ by the fifth stage of exercise. Somewhat higher cardiac out-

TABLE 1

*Cardiac output, stroke volume, heart rate and oxygen uptake responses to exercise with the McDonough and Bruce Protocol. Tests were administered pre-marathon and 1, 3, 5, and 9 days post-marathon.*

| | Pre-marathon | Post-marathon | | | |
|---|---|---|---|---|---|
| | | Day 1 | Day 3 | Day 5 | Day 9 |
| Treadmill Run Time (min:sec) | 17:45 | 15:20 | 16:23 | 17:00 | 18:20 |
| **Cardiac Output** ($\ell \cdot min^{-1}$) | | | | | |
| *Stage I | 8.83 | 7.44 | 7.42 | 5.59 | 10.21 |
| Stage II | 10.26 | 9.77 | 12.26 | 8.18 | 11.69 |
| Stage III | 18.44 | 18.01 | 22.14 | 16.27 | 14.85 |
| Stage IV | 23.36 | 24.44 | 25.87 | 22.29 | 20.77 |
| Stage V | 29.81 | — | 28.82 | 27.41 | 30.25 |
| **Stroke Volume** (ml) | | | | | |
| Stage I | 110.3 | 100.5 | 103.0 | 83.2 | 148.0 |
| Stage II | 106.9 | 106.0 | 142.5 | 99.8 | 146.0 |
| Stage III | 157.6 | 157.9 | 194.2 | 150.6 | 141.0 |
| Stage IV | 168.0 | 174.0 | 182.0 | 159.2 | 150.6 |
| Stage V | 191.1 | — | 171.5 | 169.2 | 190.0 |
| **Heart Rate** (beats$\cdot min^{-1}$) | | | | | |
| Stage I | 80 | 74 | 72 | 66 | 69 |
| Stage II | 96 | 92 | 86 | 82 | 80 |
| Stage III | 117 | 114 | 114 | 108 | 105 |
| Stage IV | 139 | 140 | 142 | 140 | 138 |
| State V | 156 | 162 | 168 | 162 | 159 |
| Max | 181 | 175 | 187 | 176 | 180 |
| **Oxygen Uptake** ($\ell \cdot min^{-1}$, STPD) | | | | | |
| Stage I | 1.12 | 0.96 | 1.02 | 0.88 | 1.11 |
| Stage II | 1.56 | 1.50 | 1.66 | 1.21 | 1.47 |
| Stage III | 2.62 | 2.41 | 2.80 | 2.28 | 2.29 |
| Stage IV | 3.75 | 3.44 | 3.57 | 3.29 | 3.19 |
| Stage V | 4.17 | 3.96 | 4.06 | 3.74 | 4.01 |
| Max | 4.20 | 3.96 | 4.10 | 3.90 | 4.40 |

*Stage I is 2.74 $km \cdot hr^{-1}$ and 10% grade, Stage II is 4.02 $km \cdot hr^{-1}$ and 12% grade, Stage III is 5.47 $km \cdot hr^{-1}$ and 14% grade, Stage IV is 6.76 $km \cdot hr^{-1}$ and 16% grade, State V is 8.05 $km \cdot hr^{-1}$ and 18% grade, Max is highest value achieved at final test stage.

put values would be expected at the final stage of exercise where maximal oxygen uptake was achieved. These cardiac output values are quite large compared to a sedentary population (Åstrand and Rodahl 1977) but consistent with data for trained middle-aged athletes (Grimby *et al* 1966). The progressive increase in cardiac output with exercise intensity appeared to be accomplished by increases in both stroke volume and heart rate. The large stroke volumes were probably a reflection of ventricular hypertrophy and increased contractility as a result of extensive training. Central circulatory dynamics did not appear to be adversely affected by competition in the marathon since cardiac output, stroke volume, and heart rate responses were similar to pre-marathon values.

The concentration of blood lactate was indicative of the anaerobic energy contribution to the exercise task. Blood lactate concentrations did not reach the pre-marathon value in response to maximal exercise at 1, 3, or 5 days post-marathon (fig. 1). Costill *et al* (1973)

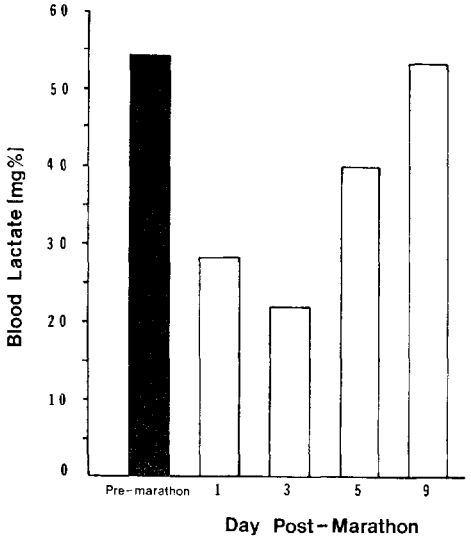


FIGURE 1. Blood lactate concentrations after maximal exercise with the McDonough and Bruce protocol (1969) pre-marathon and 1, 3, 5 and 9 days post-marathon.

observed, from tissue biopsy studies, that after exhaustive runs there was a reduced ability to produce lactate. This finding was attributed to reduced skeletal muscle glycogen stores (Costill *et al* 1973). Hermansen (1969) presented data indicating a decreased ability to produce lactate when glycogen stores fell below $1g \cdot 100g^{-1}$ muscle. Endurance exercise in excess of 2 hours at 60% of a subject's maximal oxygen uptake has been shown to deplete glycogen stores below this level. Maron *et al* (1976) reported that marathon runners work at intensities of 68–100% of their maximal oxygen uptake during competition. It can be assumed, therefore, that the subject in our study also had markedly reduced glycogen stores after the marathon.

Since skeletal muscle (Costill *et al* 1971) and hepatic (Baldwin *et al* 1975) glycogen stores were most likely decreased after the marathon, the subject was unable to mobilize this substrate. Piehl (1974) showed that the time course for refilling the glycogen stores in human muscle fibers was initially very fast when on a high carbohydrate diet, with complete refilling by 48 hours. Hultman (1967) observed a slower glycogen resynthesis (7 days) in skeletal muscles of subjects on a fat-protein diet. Our subject consumed a mixed diet post-marathon that would probably require an intermediate time for restoration of glycogen stores. By 9 days post-marathon, glycogen stores appeared to be reestablished since maximal effort exercise lactate concentration achieved the pre-marathon level.

We recognize that generalizations based on data from one subject are difficult. In the context of this study, however, we conclude that a well conditioned middle-aged runner can participate in marathon competitition with no apparent alteration in cardiovascular function during subsequent exercise and that glycogen stores used for energy metabolism require at least one week to fully recover.

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